CLINICAL PROFILE OF ST ELEVATION MYOCARDIAL INFARCTION IN FEMALES

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CERTIFICATE

This is to certify that this dissertation entitled “CLINICAL PROFILE OF ST ELEVATION MYOCARDIAL INFARCTION IN FEMALES” submitted by DR.S.HARIHARAN to The TamilNadu Dr.M.G.R. Medical University Chennai is in partial fulfillment of the regulations of the award of M.D DEGREE BRANCH 1(General medicine) and is a bonafide research work carried out by him under direct supervision and guidance.

Signature of Unit Chief

Signature of Prof.& HOD

Signature of the Dean

DECLARATION

I solemnly declare that the dissertation titled "CLINICAL PROFILE OF ST ELEVATION MYOCARDIAL INFARCTION IN FEMALES" was done by me at Stanley Medical College and Hospital during 2008-2009 under the guidance and supervision of **PROF.S.RAMASAMY, M.D.,**

The dissertation is submitted to the Tamil Nadu Dr. MGR Medical University towards the partial fulfillment of requirements for the award of M.D. DEGREE (BRANCH-I) in General Medicine.

Place: Chennai.

Date: Dr. S.HARIHARAN

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CONTENTS

INTRODUCTION

Cardiovascular disease is the leading cause of death among women, regardless of race or ethnicity, and causing the deaths of 1 in 3 women; this amounts to more deaths from heart disease than from stroke, lung cancer, chronic obstructive lung disease, and breast cancer combined.

Despite these sobering statistics and estimates that a 40-year-old woman has a lifetime risk of cardiovascular disease of 32 percent, and although awareness of cardiovascular disease as the leading cause of death has increased, still only about 55 percent of women identify cardiovascular disease as their greatest health risk.

Although mortality from heart disease has declined gradually among men since 1979 (by 30 to 50 percent), mortality from heart disease in women has increased during that same period. For coronary heart disease in specific, mortality rates have fallen for both men and women over this time period, but much more rapidly in men than women. A greater proportion of women (52 percent) than men (42 percent) with myocardial infarction die of sudden cardiac death before reaching the hospital, and two thirds of women who suffer a myocardial infarction never completely recover. Thus, understanding even subtle differences between men and women in development or progression of cardiovascular disease, use of proven therapies, and response to therapy is paramount.

Experts in industrialized societies have long recognized that the first presentation with coronary heart disease occurs approximately 10 years later among women than men, most commonly after menopause.

The worldwide *interheart Study*, a large cohort study of more than 52,000 individuals with myocardial infarction, first demonstrated that this approximate 8- to 10-year difference in age of onset among men compared with women holds widely around the world, across various socioeconomic, climatic, and cultural environments.

Although coronary artery disease in general manifests earlier in less well-developed countries, the approximate 8 to 10 year age gap in time of onset between men and women is universal. Despite this delay in onset, mortality from coronary heart disease is increasing more rapidly among women than men in both the developed and developing world.

AIM OF THE STUDY

1. To study the various presenting features of ST Elevation Myocardial Infarction (STEMI) in female pts

2. To identify important risk factors of ST Elevation Myocardial Infarction (STEMI) in our study population.

Review of literature

OVERVIEW:

Cardiovascular mortality continues at similar or increasing rates for women, whereas it is decreasing in men. Evidence based guidelines have been published following an expert panel review of the literature for the prevention of coronary artery disease in women[(1)].The importance of coronary artery disease and its prevention in women is gradually receiving increased physician and public attention. Yet, an online survey of primary care physicians, gynaecologists and cardiologists [(2)] found that fewer than 20% of physicians aware the more women than men die annually of coronary artery disease.

In addition, coronary artery disease in women is a formidable problem because of difficulties in diagnosis and increased mortality and morbidity associated with coronary artery disease events at all ages. In the middle years, coronary artery disease is associated with multiple risk factors, generally more in women than men of the same age. Intermediate-risk women, defined by Framingham criteria, were more likely to be defined as low risk by physicians surveyed in 1997.

FEMALE GENDER SPECIFIC ISSUES:

HYPERTENSION:

The prevalence of hypertension increases with advancing age; and because life expectancy is greater for women than men, there are more elderly women with hypertension [(3)]. Generally, women are more likely to have controlled blood pressure than men. However, sex

differences in autonomic nervous system may explain difficulties in blood pressure modulation in some premenopausal women when exposed to stress or vasoactive substance drugs [4].

Both systolic and diastolic blood pressures have been found in population, cohort and treatment studies to predict coronary events. Framingham data revealed that a systolic blood pressure >180, the annual incidence of coronary artery disease (angina, coronary deficiency, Myocardial Infarction or death from these diagnoses) in women older than age 65 years of age is >30%, whereas for men older than age 65 years of age, it is approximately 50 percent [5]. In other epidemiologic studies, higher diastolic blood pressure also predicts greater rates of clinical coronary artery disease [6].

Although treatment trials have also documented that lower blood pressure decreases the incidence of a first Myocardial Infarction and sudden death, this effect has been less dramatic than decrease in stroke occurrence with blood pressure control. Especially when older subjects were included in clinical trials, the benefit of treating hypertension to prevent coronary events received greater recognition.

Gender specific information about pharmacological therapy of hypertension with angiotensin-converting enzyme (ACE) inhibitors and thiazides continues to evolve.

ACE inhibitors should be used cautiously in women of reproductive age as teratogenic effects have been documented in the first as well as second trimester [7]. Potentially fertile women must understand the potential risk to the fetus before initiating therapy. Infants with only first trimester ACE inhibitor exposure had increased risk of cardiovascular and central nervous system malformations compared with infants not exposed to antihypertensive medications; other antihypertensive medications did not increase risk for congenital malformations. Cough,

common side effect of ACE inhibitors, occurred substantially more frequently in women than in men [8].

Thiazides are preferred first in the treatment of hypertension in women as well as men and are also beneficial for bone health. Epidemiological studies have documented a reduction of approximately one third in hip fracture with the use of thiazide diuretics. In randomized, double-blind, placebo trial, thiazides were associated with preservation of hip and spine bone mineral density.

LIPIDS :

There are sex difference in lipoprotein profiles and impact of lipids on cardiovascular (CV) risks [9]. Many experts consider HDL (high density lipoprotein) more predictive for women than any other lipoprotein component, with the stronger correlation between low HDL levels and coronary artery disease events.

LDL (low density lipoprotein) levels increase with increasing age for both women and men and especially important in women. In fact, one group of researchers suggest that enlarged waist (>88cm) combined with elevated triglycerides (>1.45mmol/L) best prospectively identified postmenopausal women at risk followed over 8 years [10].

Although secondary prevention with pharmacologic treatment for hyperlipidemia decreases coronary artery disease events in women as well as men, these agents are under prescribed for women after myocardial infarction and target treatment levels are often not reached. Primary prevention trials for hyperlipidemia in women with hydroxy-methylgluteryl coenzyme A (HMG- CoA) reductase inhibitors on careful review to do document evidence of

benefit with approximately 3 to 6 years of follow up, perhaps related to low events [9]. With the latest cholesterol treatment guidelines, diabetic women are candidates for primary prevention with aggressive treatment of lipid abnormalities.

DIABETES:

Diabetic individuals have higher mortality rates from coronary artery disease than non diabetics [11-!5]. In the last decade, coronary artery disease mortality rates have increased by 23 percent in diabetic women, whereas they have decreased by 27 percent in non diabetic women. This is in comparison to diabetic men, where mortality rates have declined by 13 percent; but they have decreased by 36 percent in non diabetic men.

A prospective 25 year follow up from Scotland revealed highest rates among those with both diabetes and known coronary artery disease with hazard ratios especially high for women 1.97 compared with women neither risk factor[12]. The higher coronary artery disease risk for diabetic women has been noted in multiple population studies. It has been postulated that sex differences in endothelial function, especially endothelial dependent vasodilation may play a patho physiologic role [14]. Diabetic women have coronary artery disease rates similar rates to those of diabetic men, so the female advantage is lost.

Diabetes confers substantial increased relative risk of first incident and admission for MI for women compared with age to sex matched controls younger than age 65 years over 20 years follow up in Copenhagen City heart Study [13].

Diabetic women also have higher in-hospital mortality after MI and an increased incidence of congestive heart failure (CHF) than do diabetic men. Diabetic women and men with

hypertension have especially high rates of coronary artery disease[11,15]. Women and men generally have similar incidence rates of diabetes, but more women become hypertensive with increasing age.

Lipid abnormalities are common in diabetic patients. At the time of diagnosis off type II diabetes, women have substantially lower high density lipoprotein (HDL) cholesterol than age-matched non diabetic women [17]. Other lipid abnormalities are also present, including elevated triglycerides. Subgroup analysis of diabetic diabetic patients treated with hydroxy-methylgluteryl coenzyme A (HMG- CoA) reductase inhibitors documented improved lipoprotein patterns with treatment and fewer coronary artery disease events [11].

Women at risk for developing diabetes include obese women and those who have experienced gestational diabetes. Greater weight is associated with greater insulin resistance as well as a higher rate of glucose intolerance. Even a moderate increase in physical activity and avoiding weight gain decreases the risk of developing diabetes [11].

METABOLIC SYNDROME:

The metabolic syndrome was defined in the Third Report of the National Cholesterol Education Program Expert Panel On Detection, Evaluation and Treatment of High Blood Cholesterol [18] to include obesity, glucose intolerance, hypertension and lipid abnormalities occurs in women and men at greater rates with increasing age; tobacco exposure during 12 to19 years of age dramatically increases the risk of having the metabolic syndrome [19].

Polycystic ovarian syndrome (PCOS) with increased androgens , lower HDL and higher triglycerides and higher rates of coronary artery disease may affect as many as 10 to 20 percent

of women of child bearing age[20,21]. In a retrospective chart review of PCOS, patients at an endocrine clinic also had a 43 percent prevalence of the metabolic syndrome (twice rate of metabolic syndrome for age-matched women without PCOS) once assessed [22]. Pharmacologic and lifestyle interventions may improve prognosis. Aggressive management of tobacco use, lipoprotein abnormalities and hypertension is beneficial. Regular exercise can also improve glucose and blood pressure control as well as insulin resistance [11].

OBESITY :

The prevalence of obesity has been steadily increasing with a doubling among Americans older than age 20 years from 1980 to 2002. Ethnic and racial differences in obesity are more marked among women than men. Racial differences in BMI as well as glycosylated hemoglobin start in childhood with black and Mexican American girls having less favorable profiles than white girls [23].

Obesity is linked to multiple cardiac risk factors (including insulin resistance, diabetes, hypertension and hyperlipidemia) and independently associated with coronary artery event rates [24]. The pattern of weight distribution is also predictive of coronary events, with more events among women with the apple shape, with a greater central or abdominal girth, than among those with the pear shape, with more weight in hips and buttocks. A greater waist circumference increases health risk regardless of body mass index (BMI) [25].

Increased physical activity or limited weight loss is associated with a decreased risk of coronary artery disease events. Behavioral interventions to decrease weight have been most successful when there is a physical activity component. A study of obese twins revealed the lack of physical activity correlated with the more obese twin. In the Nurses' Health Study, both BMI

>25 and physical activity were important predictors of coronary artery disease in 20 years follow up. Although new pharmacologic treatments for obesity have been developed, many have been documented to be hazardous.

PHYSICAL ACTIVITY AND EXERCISE:

Women's physiologic response to exercise includes a lower work capacity and oxygen uptake than men [(26)]. This occurs because women's cardiac output is increased by raising heart rate. Men, in comparison, accomplish an increase in cardiac output by increasing stroke volume.

Physical activity is important for primary and secondary prevention. Physical activity data collection has focused on leisure activity and excluded house work and child care. Leisure-time activity reports may greatly underestimate the actual amount of energy expended daily, especially by women.

In observing women health professionals over an average of 5 years, women who walked at least 1 hour each week had half the coronary artery disease rate as women who did not walk regularly. Both women and men benefit from referral to cardiac rehabilitation programs after MI; however, fewer women than men are referred for cardiac rehabilitation.

MENOPAUSE AND HORMONAL THERAPY:

The importance of the menstrual cycle and menopause as risk factors for coronary artery disease is still being defined. Women with early menopause after gynaecologic survey have been considered at higher risk for coronary artery disease and osteoporosis on the basis of less hormonal exposure. Although population surveys suggested hormonal therapy after menopause may decrease the risk of coronary artery disease, the women using hormones reported less

tobacco exposure, greater levels of exercise, and readier access to medical care; they also tended to be healthier and wealthier.

The Women's health initiative (WHI), a prospective randomized clinical trial of women 50 to 79 years of age, revealed the combination of estrogen and progestin after menopause increases coronary artery disease risk [(27)]; and estrogen alone in women without uterus does not decrease coronary artery disease risk. Estrogen alone in women with a uterus is contraindicated because of the associated increased risk of endometrial cancer.

In the Women's health initiative (WHI) subgroup analysis of women ages 50 to 59 years with 7-year follow up conjugated equine estrogen also was not protective. In contrast the observational Nurses' Health Study noted that women beginning hormonal therapy soon after menopause had lower coronary artery disease risk than women beginning treatment 10 years after menopause.

The earlier Heart and Estrogen Progestin Replacement Study (HERS) was the first secondary prevention clinical trial of hormonal therapy that also did not demonstrate cardiovascular benefit. These postmenopausal women had a evidence for coronary artery disease (myocardial infarction, coronary artery bypass grafting, percutaneous transluminal coronary angioplasty for occlusion >50 percent, or angiography with more than one major coronary artery).Not only was there no overall reduction in the group receiving hormonal therapy. Also remarkable in HERS was the lack of secondary prevention reported in these women with known heart disease.

National guidelines have reflected WHI and HERS results and emphasize other modalities for prevention of heart disease in women. In counseling menopausal women,

cardiovascular prevention should be a focus. Hormonal therapy is used to control severe vasomotor symptoms.

PSYCHOSOCIAL RISK FACTORS:

Both socioeconomic and psychological factors affect the prevalence and outcome of coronary artery disease. Coronary disease morbidity and mortality are greater among those of lower socioeconomic status (SES). Markers for SES have included years of formal education, owing a car, income defined by absolute or relative amount, sex, parental status and more recently race and ethnicity independent of other issues.

Depression is diagnosed twice as often in women as in men and affects outcomes in coronary artery disease. Depressive symptoms were common within the prior week in the observational arm of the WHI study, which did not include participants with major depression. History of coronary artery disease symptoms and diagnosis increased the risk of depression to a great degree than a history of a cancer. For women without coronary artery disease history followed for over 4 years, depressive symptoms adjusted for age and race was independently associated with 58 percent higher coronary artery disease mortality.

Depressive symptoms screened for at hospitalization in the Prospective Registry Evaluation Outcomes After Myocardial Infarction: Events and Recovery (PREMIER) study were common and in this cohort the depressed young women had more co morbidity and less favorable health and socioeconomic status. Only 18 percent of those depressed were discharged with anti depressant medication. Although an adequate pharmacologic interventional trial of depression diagnosed after MI in women has not been reported, selective serotonin reuptake inhibitors have been found to be clinically safe in the presence of cardiac disease.

Emotional stress, besides depression, has also been related to coronary artery disease outcomes in women. Acute and reversible cardiomyopathy has been documented after profound stress [28]. In the *Nurses' Health study*, phobic anxiety has been associated with increased risk of sudden cardiac death.

Acute and reversible severe cardiomyopathy was first described in Japan as tako-tsubo and more recently in a series of 22 women in the United States. The characteristics are the development of acute symptoms including substernal chest pain most often associated with dependent ST-segment elevation of T wave inversion of dyspnea, with profound systolic dysfunction and absence of significant luminal narrowing at angiography. Severe left ventricular systolic dysfunction and wall-motion abnormalities described as a ballooning appearance of the mid- and apical left ventricular base were documented and resolved over as little as 5 days or as long as over 2 months.

TOBACCO:

Tobacco exposure is one of the most important coronary artery disease risk factor for women and men. In epidemiologic studies greater tobacco exposure in amount and duration is related to higher coronary artery disease events in a dose-related fashion. Cigarette smoking has been associated with an earlier age of first MI and menopause. Because middle-aged women experience less symptomatic coronary artery disease than middle-aged men, the increased risk of MI and sudden death related to tobacco use is greater for women than men.

Women contemplating smoking cessation are often concerned about potential weight gain, a common sequence of efforts to stop smoking. Weight gain with tobacco cessation is on average 7 to 10 pounds, with fewer than 10 percent gaining >20 pounds. Weight gain tends to be

higher among women, blacks, and smokers who inhale >25 cigarettes per day. To avoid weight gain with tobacco cessation, several types of interventions have been recommended. Realistic expectations may be helpful as well as exercise, careful choice of snacks, and appropriate pharmacotherapy. Increasing physical activity contributes to success in smoking cessation, as does an increased expenditure of calories, even if it does not modify weight gain.

Multiple pharmacologic therapies are available. Success with nicotine replacement products is approximately double tobacco cessation compared with tobacco cessation groups alone. The patch has the highest compliance rate and provides smoother levels than the gum, spray, or lozenge. Bupropion has been found to be effective in improving tobacco cessation rates in both white and black smokers and is reported to minimize weight gain while it is used. Although bupropion is an antidepressant, it has been effective in smokers who are not depressed. Bupropion is contraindicated in patients with a history of seizures, head trauma and heavy alcohol consumption. Bupropion can exacerbate symptoms related to anorexia and bulimia and should be avoided if there is history of these disorders or recent use of a monoamine oxidase inhibitor.

Many surveys reveal the physicians can have a powerful effect on smoking cessation, even with minimal effort. Programs that promote activities to minimize weight gain and stress, social support may be more effective for women.

RACE, WOMEN AND CORONARY ARTERY DISEASE:

Racial differences in mortality, risk factors, physiology have also begun to be considered. Black women's coronary artery disease mortality rates are related to traditional coronary artery disease risk factors as well as racial and socioeconomic issues.

Combined analysis of data from the 1986 National Mortality Feedback Survey, the 1985 National Health Interview Survey, and the U.S .Bureau of the Census revealed that black women younger than age 55 years had more than twice the rate of coronary artery disease mortality as young white Americans.Coronary artery disease death rates for young black women in this study exceeds rates for young men and white women. In HERS, a large secondary prevention trial of estrogen and progestin, black women were at increased risk for coronary events at 6 years of follow up.

With the application of careful methodology, Mexican American women have coronary artery disease mortality rates higher or equal to those of non-Hispanic white American women. There are less data available on Asian Indian immigrants and those living in India, but preliminary publications report high coronary artery disease rates at a young age in both men and women.

SIGNIFICANT COMORBIDITIES:

The potential relationship between medical illness associated with inflammation and the development of coronary artery disease has been highlighted because there has been increasing attention to the potential role of inflammation as an etiology of coronary artery disease.

A case-control evaluation using population based data from *the united kingdom-based general practice research database* compared 8688 patients with mi with matched controls. Higher risk of myocardial infarction was seen in systemic lupus erythematosus(SLE) and rheumatoid arthritis (RA).

SLE (systemic lupus erythematosus):

The risk of myocardial infarction in systemic lupus erythematosus (SLE) is known to be increased at least fivefold. Coronary risk has been assessed by screening systemic lupus erythematosus (SLE) patients with electron-beam cardiac-beam CT and carotid ultrasound. When carotid ultrasound was completed on 197 systemic lupus erythematosus (SLE) patients and matched controls, carotid plaques were present among 37.1 percent of those with systemic lupus erythematosus (SLE) compared with hypertensive controls. SLE patients with carotid plaques were generally older than age 40, with a longer duration of SLE and less likely to be treated with prednisone, cyclophosphamide or hydroxychloroquine.

Rheumatoid arthritis:

Rheumatoid arthritis has also been associated with increased cardio vascular mortality and morbidity. Coronary artery disease may occur a decade earlier. Assessment for preclinical evidence of atherosclerosis among 98 women with rheumatoid arthritis through carotid ultrasonography documented carotid atherosclerosis in 44 percent of those with rheumatoid arthritis compared to 15 percent of age matched hypertensive controls. Methotrexate, which decreases the inflammatory process in rheumatoid arthritis, is also associated with lower rates of coronary artery disease.

INTERVENTIONS FOR CORONARY ARTERY DISEASE:

Gender differences in the prevalence and complication of percutaneous coronary interventions (PCI) and CABG (coronary artery bypass grafting) surgery are evolving. There is controversy whether women are undertreated or men are over treated.

Currently approximately 33 percent of the PCIs (Percutaneous Coronary Interventions) are performed in women. Primary angiography is superior to fibrinolytic therapy when available within 3 hours of symptoms, even with the higher procedure complication rate for women. The development of smaller coronary artery catheters only partially decreased the sex difference in complications. Most experts note that the higher short and long-term complication rates in women are probably also related to older age, more co morbidities, and longer duration of symptoms compared to men. Vascular complications at the access site, retroperitoneal or requiring transfusion are more common in women than men [(29)].

In a clinical trial comparing PCI balloon versus stenting with or without the addition of abciximab, overall women received treatment after a longer delay and a higher 1-year mortality, revascularization, and adverse cardiac events. Women benefitted most from stenting with lower revascularization and adverse events. In women, less vascularization in the first 30 days was required without an increased bleeding risk with the addition of abciximab, but no difference was found at 1 year. CABG (Coronary Artery Bypass Grafting) surgery is also more commonly performed in men than women [(29)].

Experience over a decade with off-pump CABG revealed women were older and had a smaller surface area. After controlling for these differences and other co morbidities, women had more wound infections and longer hospitalization but similar mortality.

Among patients at least 75 years old at the time of CABG in the Society of Thoracic Surgeons' voluntary data base, women were 38.9 percent of the sample and were older with smaller body surface area. Women were more likely to have emergency surgery and had higher operative mortality, more pulmonary and vascular complications, and longer hospitalizations [(30)].

SUDDEN DEATH:

In the Nurses' Health Study cohort, 636 women or 57.3 percent of the cohort died out of the hospital or in the emergency room. There was no prior history of coronary artery disease in 69 percent of the women with sudden death. At least one coronary risk factor was present in 94 percent of those who died. The strongest associations were with cigarette smoking, diabetes, and hypertension [31].

The Nurses' Health Study assessed phobic anxiety, which is more common in women than men, and found it correlated with higher smoking rates, hypertension, diabetes, hypercholesteremia, and BMI >30kg/m2. Similar to prior results in men, high levels of phobic anxiety in women were associated with an increased risk of fatal coronary artery disease including sudden death, even after controlling for these co morbidities in women with no coronary artery disease initially. Out-of-hospital arrest survival is similar for women and men; however men had higher rates of ventricular fibrillation.

ARRHYYTHMIAS:

Women generally have higher heart rate than men and respond to increasing cardiac demand by increasing heart rate rather than increasing stroke volume [26]. Women in their seventh decade with a heart rate >77 beats/min had substantially higher cardio vascular and all-cause mortality rates than women with slower pulses with 6 years of follow up after controlling for potential confounders such as prior coronary artery disease, smoking, hypertension, activity level and anemia.

Gender differences in the incidence and management of arrhythmias have been noted. There is a female predominance of atrio ventricular node reentrant tachycardia and orthodromic

supraventricular tachycardia, but men are likely to have atrial and ventricular fibrillation associated with wolff-parkinson-white syndrome. Although in the treatment of supra ventricular reentrant tachycardias there has been increasing use of radiofrequency catheter ablation therapy, limited data has focused on gender differences.

Overall, women have a higher risk of dying of atrial fibrillation. In an important clinical randomized control trial comparing rate and rhythm control in patients with atrial fibrillation despite prior electrical cardioversion, the primary endpoints (composite of death from coronary artery disease, heart failure, thromboembolism complications, bleeding, the need for pacemaker implantation or severe adverse effects of antiarrhythmic agents) had important sex differences.

For women, rhythm control had substantially worse outcomes with an absolute differences of 21.5 compared with results for men revealing only a trend with absolute difference of 3.9. Therefore for women, it may be beneficial to focus on rate rather than rhythm control in persistent atrial fibrillation.

Drug-induced torsade de pointes occurs more commonly in women than men [(8)]. As this sex difference is noticed after puberty, a prospective study explored the impact of the menstrual cycle on the development of torsade de pointes. After infusion of low dose ibutilide, the greatest increase in QT interval was seen in women during menses and in their ovulatory phase compared with women during luteal phase of menstruation and men.

DIAGNOSIS OF CORONARY ARTERY DISEASE IN WOMEN:

Once coronary artery disease is considered as a diagnosis, further evaluation is required to assess disease presence and severity. Exercise stress testing was the earliest noninvasive way to assess coronary artery disease risk. However, women have lower sensitivity and specificity

with exercise stress testing than men, in part related to lower ecg voltage. In multiple populations women have been noted to have more frequent ST-T-wave changes [32]. Gender-specific criteria for exercise stress testing interpretation have been proposed to compensate for generally smaller ST-segment changes seen in women.

After exercise stress testing, the maximal exercise capacity and heart rate recovery in women are important prognostically. Among asymptomatic women with low Framingham scores, those with lower exercise capacity and slower heart rate recovery were at increased risk for CV death.

Stress imaging techniques are often required in assessing symptomatic women for severity of coronary artery disease. Nuclear stress perfusion testing in women can be potentially hindered by soft tissue attenuation from breast tissue with the use of thallium, so technetium may be preferred. Stress echocardiography is highly dependent on operator expertise and may be technically difficult in obese patients.

Because cardiac catheterization is less likely to reveal coronary artery disease in women, many clinicians initially evaluate women at intermediate risk with stress-imaging techniques. For example, anginal symptoms are less predictive of abnormal coronary anatomy in women than men. Direct referral to cardiac catheterization should occur with a high suspicion of significant coronary artery disease that might benefit from intervention or after an abnormal noninvasive stress test.

ST Elevation Myocardial Infarction (STEMI) - Pathology, clinical features and management:

The classic World Health Organization (WHO) criteria for the diagnosis of MI require that at least two of the following three elements be present: a history of ischemic-type chest discomfort, evolutionary changes on serially obtained ECG tracings, and a rise and fall in serum cardiac markers [33].

Revised Definition of Myocardial Infarction (MI) [34]

A. Criteria for Acute, Evolving, or Recent MI:

1. Typical rise and/or fall of biochemical markers of myocardial necrosis with at least one of the following

A) Ischemic symptoms
B) Development of pathological Q waves in the ECG
C) ECG changes indicative of ischemia (ST segment elevation or depression)
D) Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

2. Pathological findings of an acute myocardial infarction.

(Either of the above criteria satisfies the diagnosis for acute, evolving or recent MI)

B. Criteria for Healing or Healed Myocardial Infarction:

1. Development of new pathological Q waves in serial ECGs. The patient may or may not remember previous symptoms. Biochemical markers of myocardial necrosis may have normalized depending on the length of time that has passed since the infarction developed.

2. Pathological findings of a healed or healing infarction.

(Any one of the above criteria satisfies the diagnosis for healing or healed MI)

Pathology :

Almost all myocardial infarctions result from coronary atherosclerosis, generally with superimposed coronary thrombosis.

Plaque :

During the natural evolution of atherosclerotic plaque, especially that which is lipid laden, an abrupt and catastrophic transition can occur, characterized by plaque disruption. Some patients have a systemic predisposition to plaque disruption that is independent of traditional risk factors.

Plaque disruption exposes substances that promote platelet activation and aggregation, thrombin generation, and ultimately thrombus formation [(35)]. The resultant thrombus interrupts blood flow and leads to an imbalance between oxygen supply and demand and, if this imbalance is severe and persistent, to myocardial necrosis .

Composition of plaques :

At autopsy, the atherosclerotic plaques of patients who died of STEMI are composed primarily of fibrous tissue of varying density and cellularity with superimposed thrombus. Calcium, lipid-laden foam cells, and extracellular lipid each constitute 5 to 10 percent of the remaining area. Coronary arterial thrombi responsible for STEMI are approximately 1cm in length in most cases; adhere to the luminal surface of an artery; and contain platelets, fibrin, erythrocytes, and leukocyte. The composition of the thrombus may vary at different levels: A white thrombus is composed of platelets, fibrin, or both, and a red thrombus is composed of erythrocytes, fibrin, platelets, and leukocytes. Early thrombi are usually small and nonocclusive and are composed predominantly of platelets.

Causes of Myocardial Infarction without Coronary Atherosclerosis [36] :

1. Coronary artery disease Other than Atherosclerosis

-Arteritis

Luetic

Granulomatous (Takayasu disease)

Polyarteritis nodosa

Mucocutaneous lymph node (Kawasaki) syndrome

-Trauma to coronary arteries

-Coronary mural thickening with metabolic disease or intimal proliferative disease (Hurler, Fabry disease, Homocystinuria, Amyloidosis)

2. Emboli to Coronary Arteries

3. Congenital Coronary Artery Anomalies

4. Miscellaneous (cocaine abuse, myocardial contusion, Polycythemia vera)

Risk Factors for Atherothrombotic Diseases in Women:

Smoking

Hypertension

Hyperlipidemia and Elevated Low-Density Lipoprotein Cholesterol

The metabolic syndrome

Insulin resistance

Obesity

Diabetes

Polycystic ovary disease

Age >55yr

Inactivity

Homocysteine elevations

Family history of premature coronary death: younger than 55 yr in men and 65 yr in women (first degree relative)

Lipoprotein(a)

Oral contraceptive use in the presence of other risk factors

CLINICAL FEATURES:

1. **Chest pain :**

The pain of STEMI varies in intensity; in most patients, it is severe and in some instances intolerable. The pain is prolonged, usually lasting for more than 30 minutes and frequently for a number of hours.

The discomfort is described as constricting, crushing, oppressing, or compressing; often the patient complains of a sensation of a heavy weight or a squeezing in the chest. Although the discomfort is typically described as a choking, viselike, or heavy pain, it can also be characterized as a stabbing, knifelike, boring, or burning discomfort. The pain is usually retrosternal in location, spreading frequently to both sides of the anterior chest, with predilection for the left side.

2. OTHER SYMPTOMS:

Nausea and vomiting may occur, presumably because of activation of the vagal reflex or stimulation of left ventricular receptors as part of the Bezold-Jarisch reflex. These symptoms occur more commonly in patients with inferior STEMI than in those with anterior STEMI. Moreover, nausea and vomiting are common side effects of opiates.

When the pain of STEMI is epigastric in location and is associated with nausea and vomiting, the clinical picture can easily be confused with that of acute cholecystitis, gastritis, or peptic ulcer. Occasionally, a patient complains of diarrhea or a violent urge to defecate during the acute phase of STEMI. Other symptoms include feelings of profound weakness, dizziness, palpitations, cold perspiration, and a sense of impending doom.

3. SILENT STEMI:

Unrecognized or silent infarction occurs more commonly in patients without antecedent angina pectoris and in patients with diabetes and hypertension [37]. Silent STEMI is often followed by silent ischemia. The prognoses of patients with silent and symptomatic presentations of STEMI appear quite similar.

4. Atypical presentations of STEMI:

(1) Heart failure (i.e., dyspnea without pain beginning de novo or worsening of established failure);

(2) Classic angina pectoris without a particularly severe or prolonged episode;

(3) Atypical location of the pain;

(4) Central nervous system manifestations, resembling those of stroke, secondary to a sharp reduction in cardiac output in a patient with cerebral arteriosclerosis; (5) apprehension and nervousness;

(6) Sudden mania or psychosis;

(7) Syncope;

(8) Overwhelming weakness;

(9) Acute indigestion;

(10) Peripheral embolization.

Laboratory findings:

1. Serum Markers of Cardiac Damage :

i. **CREATINE KINASE(CK) :**

Serum CK activity exceeds the normal range within 4 to 8 hours after the onset of STEMI and declines to normal within 2 to 3 days. Although the peak CK occurs on average at about 24 hours, peak levels occur earlier in patients who have had reperfusion as a result of the administration of fibrinolytic therapy or mechanical recanalization (as well as in patients with early spontaneous fibrinolysis). Because reperfusion influences the time-activity curve of serum CK, and because reperfusion itself influences infarct size, reperfusion confounds estimation of infarct size by enzyme analysis [(38)].

ii. **CREATINE KINASE ISOENZYMES:**

Creatine kinase MB is analyzed in most laboratories by highly sensitive and specific enzyme immunoassays that utilize monoclonal antibodies directed against CK-MB [(39)]. Mass assays report results in nanograms per milliliter rather than units per milliliter and have been confirmed to be more accurate than CK-MB activity assays, especially in patients presenting within 4 hours of the onset of STEMI.

It has been proposed that a ratio (relative index) of CK-MB mass to CK activity of about 2.5 is indicative of a myocardial rather than a skeletal source of the CK-MB elevation.

Although this ratio may be satisfied by many patients with STEMI, it is inaccurate in several circumstances: (1) when high levels of total CK are present because of skeletal muscle injury (a large quantity of CK-MB must be released from the myocardium to satisfy criteria); (2) when chronic skeletal muscle injury releases large amounts of CK-MB; and (3) when total CK

measurements are within the normal reference range for the laboratory and CK-MB is elevated (possibly indicating that a microinfarction has occurred). Patients with minimally elevated CK-MB and normal CK have a prognosis that is generally worse than that for patients with suspected MI but no CK-MB elevation.

iii. CARDIAC-SPECIFIC TROPONINS:

Although both TnT and TnI are present in cardiac and skeletal muscle, they are encoded by different genes and their amino acid sequence differs. This permits the production of antibodies that are specific for the cardiac form (cTnT and cTnI) and has led to the development of quantitative assays for cTnT and cTnI that have been approved by the Food and Drug Administration for clinical use.

Several studies have confirmed the reliability of these new quantitative assays for detecting myocardial injury, and measurement of cTnT or cTnI is now at the center of the new diagnostic criteria for MI.

In patients with MI, cTnT and cTnI first begin to rise above the upper reference limit by 3 hours from the onset of chest pain. Because of a continuous release from a degenerating contractile apparatus in necrotic myocytes, elevations of cTnI may persist for 7 to 10 days after MI; elevations of cTnT may persist for up to 10 to 14 days. The prolonged time course of elevation of cTnT and cTnI is advantageous for the late diagnosis of MI. Patients with STEMI who undergo successful recanalization of the infarct-related artery have a rapid release of cardiac troponins that also may be useful as an indicator of reperfusion.

Troponin Versus CK-MB:

When comparing the diagnostic efficiency of the cardiac troponins versus CK-MB for MI, it is important to bear in mind that the troponin assays can probably detect episodes of myocardial necrosis that are below the detection limit of the current CK-MB assays, leading to a number of "false-positive" cases of troponin elevations if CK-MB is used as the reference standard or, conversely, false-negative cases of CK-MB elevation if troponin is used as the reference standard.

RECOMMENDATIONS FOR MEASUREMENT OF SERUM MARKERS:

It seems reasonable for clinicians to measure either cTnT or cTnI in all patients with suspected MI. From a cost-effectiveness perspective, it is unnecessary to measure both a cardiac-specific troponin and CK-MB. Routine diagnosis of MI can be accomplished within 12 hours using CK-MB, cTnT, or cTnI by obtaining measurements approximately every 8 to 12 hours.

Retrospective diagnosis or diagnosis of MI in the presence of skeletal muscle injury is more readily accomplished with cTnT or cTnI. With increasing familiarity of clinicians with assays for the cardiac-specific troponins, it is anticipated that they will supersede assays for CK-MB not only for the diagnosis of MI but also for assessment of reperfusion, reinfarction, and estimation of infarct size [40].

MYOGLOBIN:

Peak levels of serum myoglobin are reached considerably earlier (1 to 4 hours) than peak values of serum CK. Because of its lack of cardiac specificity, an isolated measurement of myoglobin within the first 4 to 8 hours after the onset of chest discomfort in patients with a

nondiagnostic ECG should not be relied on to make the diagnosis of MI but should be supplemented by a more cardiac-specific marker such as cTnI or cTnT.

SERUM LIPIDS:

During the first 24 to 48 hours after admission, total cholesterol and high-density lipoprotein (HDL) cholesterol remain at or near baseline values but generally fall precipitously after that. The fall in HDL cholesterol after STEMI is greater than the fall in total cholesterol; thus the ratio of total cholesterol to HDL cholesterol is no longer useful for risk assessment unless measured early after MI. A lipid profile should be obtained on all STEMI patients who are admitted within 24 to 48 hours of symptoms.

HEMATOLOGICAL FINDINGS:

The elevation of the white blood cell count usually develops within 2 hours after the onset of chest pain, reaches a peak 2 to 4 days after infarction, and returns to normal in 1 week; the peak white blood cell count usually ranges between 12 and 15× 10^3/ml but occasionally rises to as high as 20× 10^3/ml in patients with large STEMI.

The erythrocyte sedimentation rate (ESR) is usually normal during the first day or two after infarction, even though fever and leukocytosis may be present. It then rises to a peak on the fourth or fifth day and may remain elevated for several weeks. The increase in the ESR does not correlate well with the size of the infarction or with the prognosis.

The hemoglobin value at presentation with STEMI powerfully and independently predicts major cardiovascular events [41].

Electrocardiography (ECG):

Although general agreement exists on electrocardiographic and vectorcardiographic criteria for the recognition of infarction of the anterior and inferior myocardial walls, less agreement pertains to criteria for lateral and posterior infarcts; in this area, even the terminology can be confusing. A consensus group has recommended elimination of the term “posterior” and suggests using “lateral” to be consistent with current understanding of the segmental anatomy of the heart as it sits in the thorax.

Patients with an abnormal R wave in V_1 (0.04 second in duration and/or R/S ratio ≥1 in the absence of preexcitation or right ventricular hypertrophy) with inferior or lateral Q waves have an increased incidence of isolated occlusion of a dominant left circumflex coronary artery without collateral circulation; such patients have a lower ejection fraction, increased end-systolic volume, and higher complication rate than patients with inferior infarction because of isolated occlusion of the right coronary artery.

Q-WAVE AND NON-Q-WAVE INFARCTION:

As noted earlier, the presence or absence of Q waves on the surface ECG does not reliably distinguish between transmural and nontransmural (subendocardial) MI [(42)]. Q waves on the ECG signify abnormal electrical activity but are not synonymous with irreversible myocardial damage. Also, the absence of Q waves may simply reflect the insensitivity of the standard 12-lead ECG, especially in zones of the left ventricle supplied by the left circumflex artery .

Angiographic studies in MI patients without ST-segment elevation show a higher incidence of subtotal occlusion of the culprit coronary vessel and greater collateral flow to the infarct zone. Observational data suggest that MI without ST-segment elevation occurs more commonly in elderly patients and patients with a prior MI.

Chest roentgenogram :

The degree of congestion and the size of the left side of the heart on the chest film are useful for defining groups of patients with STEMI who are at increased risk of dying after the acute event.

Echocardiography:

The relative portability of echocardiographic equipment makes this technique ideal for the assessment of patients with MI hospitalized in the coronary care unit or even in the emergency department before admission.

In patients with chest pain compatible with MI but with a nondiagnostic ECG, the finding on echocardiography of a distinct region of disordered contraction can be helpful diagnostically because it supports the diagnosis of myocardial ischemia.

Areas of abnormal regional wall motion are observed almost universally in patients with MI, and the degree of wall motion abnormality can be categorized with a semiquantitative wall motion score index. Of note, abnormal wall motion is less often noted echocardiographically when the infarction is small and the age of regional wall motion abnormality cannot always be determined.

Left ventricular function estimated from two-dimensional echocardiograms correlates well with measurements from angiography and is useful in establishing prognosis after MI [(43)]. Furthermore, the early use of echocardiography can aid in the early detection of potentially viable but stunned myocardium (contractile reserve), residual provocable ischemia, patients at risk for the development of congestive heart failure after MI, and mechanical complications of MI.Management:

i. Management in the Emergency Department: the goals are

- Control of cardiac discomfort

- Rapid identification of patients who are candidates for reperfusion therapy

- Triage of lower-risk patients to appropriate location in the hospital

-Avoidance of inappropriate discharge of patients with STEMI

-Aspirin (160 mg-325 mg)

-Nasal O2 if when hypoxemia is present

ii. Control of Discomfort:

i.Sublingual nitroglycerin :

Up to three doses of 0.4 mg should be administered at about 5-min intervals. By virtue of their ability to enhance coronary blood flow by coronary vasodilation and to decrease ventricular preload by increasing venous capacitance, sublingual nitrates are indicated for most patients with an acute coronary syndrome. At present, the only groups of patients with STEMI in whom sublingual nitroglycerin should *not* be given are those with inferior MI and suspected right ventricular infarction or marked hypotension (systolic pressure <90 mm Hg), especially if accompanied by bradycardia.

Intravenous Morphine of 2-4mg:

The reduction of anxiety resulting from morphine diminishes the patient's restlessness and the activity of the autonomic nervous system, with a consequent reduction of the heart's metabolic demands. The beneficial effect of morphine in patients with pulmonary edema is unequivocal and may be related to several factors including peripheral arterial and venous dilation (particularly among patients with excessive sympathoadrenal activity), reduction of the

work of breathing, and slowing of heart rate secondary to combined withdrawal of sympathetic tone and augmentation of vagal tone.

Intravenous beta blockers:

These drugs relieve pain, reduce the need for analgesics in many patients, and reduce infarct size and life-threatening arrhythmias. Avoiding early intravenous blockade in patients presenting in Killip Class II or greater is important, however, because of the risk of precipitating cardiogenic shock.

A popular and relatively safe protocol for the use of a beta blocker in this situation is as follows

. (1) Patients with heart failure (rales >10 cm up from diaphragm), hypotension (blood pressure <90 mm Hg), bradycardia (heart rate <60 beats/min), or heart block (PR interval >0.24 sec) are first excluded.

(2) Metoprolol is given in three 5-mg intravenous boluses.

(3) Patients are observed for 2 to 5 minutes after each bolus, and if the heart rate falls below 60 beats/min or systolic blood pressure falls below 100 mm Hg, no further drug is given.

Management:

The primary tool for screening patients and making triage decision is the initial 12-lead ECG. When ST-segment elevation of at least 2mm in two contagious precordial leads and 1mm in two contagious limb lead is present , a patient should be considered a candidate for reperfusion therapy.

The process of selecting patients for fibrinolysis versus PCI (percutaneous coronary intervention) is discussed below. In the absence of ST-segment elevation, fibrinolysis is not helpful, and evidence exists suggesting that it may be harmful.

Reperfusion Options for STEMI Patients [44]:

Step 1: Assess time and risk:

- Time since onset of symptoms

- Risk of STEMI

- Risk of fibrinolysis

- Time required for transport to a skilled PCI laboratory

Step 2: Determine if . brinolysis or invasive strategy is preferred

- If presentation is <3 hr and there is no delay to an invasive strateg y, there is no preference for either strategy.

Fibrinolysis is generally preferred if:

i. Early presentation (≤3 hr from symptom onset and delay to invasive strategy)

ii. Invasive strategy is not an option:

- Catheterization laboratory occupied or not available

-Vascular access difficulties

- Lack of access to a skilled PCI laboratory

- Delay to invasive strategy (delay in transport)

An invasive strategy is generally preferred if [44]:

- Skilled PCI lab is available with surgical backup

-High risk from STEMI

- Cardiogenic shock

- Killip class ≥3

- Contraindications to fibrinolysis including increased risk of bleeding, ICH

-Late presentation

- Symptom onset was more than 3 hr ago

-Diagnosis of STEMI is in doubt.

Contraindications and Cautions for Fibrinolytic Use in STEMI [44]:

Absolute Contraindications:

- Any prior intracranial hemorrhage
- Known structural cerebral vascular lesion (e.g., arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 mon except acute ischemic stroke within 3 hr
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed head or facial trauma within 3 mo

Relative contraindications:

- Severe uncontrolled hypertension on presentation (SBP >180 Hg or DBP >110 Hg)
- History of prior ischemic stroke >3 mo dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (>10 min) CPR or major surgery (<3 wk)
- Recent (within 2-4 wk) internal bleeding
- For streptokinase/anistreplase: prior exposure (>5 days ago) or prior
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

MATERIALS AND METHODS

The study was conducted at intensive cardiac care unit of Govt. Stanley Hospital, Chennai during July 09 to November 09.

- This study was done as a descriptive study.
- Ninety female patients admitted with clinical features and ECG changes suggestive of ST segment elevation myocardial infarction, elevated CK-MB taken as cases.
- Inclusion criteria:

  - Female patients admitted with the clinical features and ECG changes suggestive of STEMI.

- Exclusion criteria:

  - Female patients with unstable angina, NSTEMI (Non STEMI)

  -Female patients with ST elevation without both symptoms and enzyme elevation.

- Ninety patients were examined and detailed history with regard to history risk factor analysis was made.
- Baseline investigations like complete blood count, renal function test, Urine routine examination, Chest x ray and ECG were taken.

- In this study, clinical symptoms like chest pain and association with sweating, palpitation, nausea, vomiting and breathlessness were all taken into account and percentage of each was studied.

- Physical signs like hypertension, hypotension, raised JVP, S3, S4, crackles and wheeze were all looked for.

- Hypertension was considered by documentary history of hypertension on medication or BP >140/90 mmHg.

- Diabetes mellitus was considered either by documentary history of treatment or fasting blood sugar >126mg/dl and postprandial blood sugar >200mg/dl.

- Serum cholesterol was done for all patients and lipid profile was also done.

LDL, HDL and triglycerides levels were taken into the study.HDL level was graded as >50mg/dl, 40-50mg/dl ,40-30mg/dl,30-20mg/dl and <20mg/dl.LDL levels should be ideally <130 mg/dl and LDL level was graded as >130mg/dl,130-160mg/dl and >160 mg/dl.

- Patients were grouped into four categorized according to their age as <45yrs,45 -60 yrs, 60-75yrs and >75 yrs.

- A detailed family history, socioeconomic status, occupation, menstrual history, diet and lifestyle were obtained.

- Killip score is calculated based on classification according to the status of cardiac pump function , estimated clinically

Class I – No signs of pulmonary congestion or shock

Class II – Moderate heart failure as evident by rales at the lung bases, S3 gallop, tachypnoea

Class III- Severe heart failure (pulmonary edema)

Class IV- Cardiogenic shock with systolic BP < 90 mmHg, peripheral Vasoconstriction, cyanosis, oliguria and confusion

- Body Mass Index (BMI) was calculated by using Weight in kilogram/height^2 in metre^2.
- Type of myocardial infarction was based on surface ECG

Type of Myocardial Infarction(MI)	ECG pattern
Anterior wall MI(extensive)(AWMI)	Typical infarction pattern in lead I,aVL,V1-V6
Antero septal MI(ASMI)	Infarction pattern in lead V1-V4
Lateral Wall(Apical) MI(LWMI)	Infarction pattern in lead V5,V6
Anterolateral wall MI	Lead I,aVL,V4-V6
Inferior Wall MI(IWMI)	Lead II,III,aVF
IWMI + Right Ventricular MI	Lead II,III,aVF+ Righted sided lead V4R
IWMI + RWMI + Posterior Wall MI	Lead II,III,aVF + lead V4R +mirror image classical pattern in V1-V3
Combined. others	Depends on type of involvement

OBSERVATIONS

Clinical presentation-Symptoms:

- 79% (71 out of the 90 patients) of the patients with ST elevation MI had mild to severe chest pain. The character of the pain was assessed to be squeezing or compressing in nature with characteristic radiation in 27% of the 71patients.
- 21% of the patients (19 out of the 90 patients) presented only with breathlessness without chest pain.
- One patient presented with cerebro vascular accident(CVA)

Table 1

Clinical presentation

Symptoms	No. of Cases(total cases=90)
Chest pain(mild to severe angina)	71
Radiation	19
Vomiting	15
Nausea	17
Breathlessness	29
Breathless without Chest pain	19
Palpitation	22
Epigastric pain	12
Others (CVA, Asymptomatic)	1

In our study, out of 90 patients, 19 patients were presented without typical chest pain.

Table 2

Age wise distribution of patients without chest pain:

Age	No. of patients
<45 yrs	0
45-60	2
60-75	7
>75yrs	10
Total	19

Age wise distribution of patients without chest pain:

Table 3

Prevalence of Diabetes in patients presented without chest pain:

Diabetic status	No. of patients
Diabetic	12
Non-diabetic	7
Total	19

CLINICAL PRESENTATION – SIGNS:

- Basal crackle is the most common presenting sign. Jugular venous pressure is elevated in 7 patients.
- Basal crackles were found in 36% (32 out of the 90)patients.
- Three patients were in hypotension and six patients were admitted with mitral regurgitation.

Table 4

Signs	No. of cases (total 90)
Hypertension	27
Hypotension	3
Crackles	32
Raised JVP	12
Associated MR	6
Pedal edema	18
Apical shift	13
Wheeze	7
S3	21
S4	5
Pallor	24

Killip Classification:

In our study, most of the patients presented with Killip Class I. Out of the 90 patients, 58 patients presented with Killip class I and 20 patients were in Class II.

Table 5

Number of patients according to Killip Classification:

Class	No of cases(Total=90)
Class I	58
Class II	20
Class III	9
Class IV	3

Thrombolysis:

In our study, 48 patients (53%) were thrombolysed and 42 patients (47%) were not thrombolysed

Incidence of non-thrombolysis due to various reasons increases with the age.55% of female with age 75 and above were not thrombolysed, compared to 40% in women in age 60 and less than 60 yrs.

Table 6

Age distribution	Thrombolysed	Not thrombolysed
Age <45yrs	2	2
Age 45-60yrs	16	10
Age 60-75 yrs	16	13
Age >75 yrs	14	17
Total	48	42

Age:

Incidence of myocardial infarction increases with the age. In our study 67% of the patients were in the age of >60yrs.

Table 7

Age wise distribution of patients(Total-90)

Age Distribution	No. of the patients
Age <45yrs	4
Age 45-60yrs	26
Age 60-75 yrs	29
Age >75 yrs	31

Diabetes :

In our study, 58% population(52 out of the 90 patients) found to have diabetes.

Table 8

Diabetic status in study population

Diabetic	52
Non Diabetic	38

Diabetic status:

Hypertension:

In our study, 40% population (36 out of the 90 patients) found to have hypertension.

Dyslipidemia:

Total cholesterol was measured for all patients. Lipid profiles mainly LDL, HDL were measured. Out of the 90 patients, 81 patients were found to have dyslipidemia.

LDL Cholesterol level:

Table-9

HDL Cholesterol level	No. of Patients
<20 mg/dl	7
20-30 mg/dl	37
30-40 mg/dl	27
40-50 mg/dl	14
>50 mg/dl	5

HDL Cholesterol:

Table-10

LDL Cholesterol	No. of patients
<130 mg/dl	16
130-160 mg/dl	23
>160 mg/dl	51

Obesity:

Obesity is an independent risk factor for increased mortality. In our study, 59% percent (26 patient in Class I Obese, 22 in Class II Obese and 3 in Morbid Obese) were in obese category and 29% percent(26 patient) were in overweight category.

Table -11

Classification of Overweight and Obesity by BMI for Indians:

	Obesity class	BMI (kg/m2)	No. of patients
Underweight		<18.5	0
Normal		18.5-22.9	9
Overweight		23.0-27.9	26
Obesity	Class I	28.0-32.9	29
Obesity	Class II	33.0-37.9	23
Extreme Obesity	Class III	>38	3

Alcohol and Smoking:

Prevalence of smoking and alcohol in our study was zero percent..

Type of Myocardial Infarction :

In our study, Anteroseptal and anterior wall myocardial infarction were the two common type of myocardial infarction.

Table-12

Type of Myocardial Infarction(MI)	No. of patients
ASMI	26
AWMI	17
ALMI	10
Apical(lateral)MI	3
IWMI	19
IWMI+RVMI	11
IWMI+RVMI+PWMI	4

Total cases = 90 and Distribution of cases depends on surface ECG:

Discussion

Women constitute 48% of the total population in India, However, due to inadequate perception and attention, coronary heart disease remains a formidable health problem in women.

The lifetime risk of a woman dying from heart disease is more than eight-times than from breast cancer, yet they fear more for breast cancer and neglect any precautions for heart diseases. It is estimated that 31% of women will die from coronary artery disease; yet, about 70% of university educated women consider their risk of coronary artery disease to be <1%.

In our study, 71 out of the 90 patients (79%) had typical angina symptoms. The other two common symptoms in our study, were breathlessness and palpitation

In our study, 21% (19 out of the 90 patients) of patients were presented without chest pain and 18% of patients were presented with atypical symptoms like vomiting, nausea and epigastric pain. Out of those 19 patients, 10 patients were in the age group of >75 years and 12 patients were diabetic.

In a study by **Marrugat et al.,(1998)** showed that women are more likely to have symptoms such as nausea, instead of chest pain.

In our study, 67% of patients were in the age group of above 60yrs. Coronary artery disease mortality among women gradually increases with age and increase in the

risk of coronary artery disease is related to a higher incidence of hypertension, diabetes, dyslipidemia, and obesity.

In our study, 58% were found to have diabetes. Cardiovascular disease is the most common complication of diabetes in women, and diabetes is the only condition that causes women to have rates of coronary artery disease similar to those of men.

Huxley and colleagues found women with diabetes had 3.5 fold increase in cardiovascular mortality compared with non-diabetic women as well as their male counterpart.

Diabetes confers substantial increased relative risk of first, incident, and admission for MI for women compared with age-to-sex matched controls younger than age 65 yrs over 20 yrs follow up in **Copenhagen City Heart Study**[13].

In our study, 40 % were found to have hypertension. Having hypertension raises a woman's risk of heart disease four fold, and her risk of dying from a coronary event seven-fold. Besides the usual factors-salt intake, inactivity, age, and genetics-hypertension in women is particularly closely correlated with obesity.

In our study, prevalence of smoking and alcohol was zero percent and India is fortunate in that like other Asian countries only few women smoke, which is lower than many western countries. But even then they are not saved from the harmful effects of passive smoking, as their male counterparts smoke with high prevalence of 60%.

A study by Liaquat Ali Cheema et al., (Gender comparison of coronary risk factors and clinical presentation in Pakistani patients with coronary artery lesions):

This showed that smoking was not a risk factor in females in the study population and diabetes mellitus was more common in females while smoking and dyslipidemia in males.

H Fu and Y Zhao study*:* (study of risk factors for female patients with acute myocardial infarction,2009)

Five hundred and eighty female patients were compared with 2,058 male patients for age, occupation, positive family history, type 2 diabetes mellitus, hypertension, and hyperlipemia.

Compared with male patients, female patients with AMI were older, and type 2 diabetes mellitus and hypertension probably played more important roles in female patients.

In a study by V. Chiamvimonvat and L. Sternberg(University of Toronto):

Coronary artery disease is the leading cause of mortality in women, with incidence after menopause equal to that of men. Diabetes and postmenopausal status without hormone replacement therapy are the strongest risk factors.

Women with myocardial infarction are more likely to have atypical symptoms, including nonexertional chest pain; pain in other locations, such as jaw, arms, shoulder,

back, and epigastrium; and angina-equivalents, such as dyspnea, palpitations, and presyncope.

Halvorsen S and Risoe C. study: (Symptoms and diagnosis of coronary heart disease in women)

Myocardial infarction hits women about ten years later than men, but women with risk factors lose their sex advantage. Women with coronary disease are less likely to experience chest pain than men and may have less specific symptoms.

Mosca et al study:

In this study, compared with male patients, female patients have 41% identified being overweight,, 36% said smoking, 31% cited high cholesterol, 29% identified family history, 19% said hypertension, 19% identified diabetes and 1% said high triglyceride level (Mosca et al., 2004).

In our study, 29% of patients were in overweight category(BMI=>23 to <27.9) and 61% were obese(BMI >28). Obesity is linked to multiple cardiac risk factors (including insulin resistance, diabetes, hypertension, and hyperlipidemia) and independently associated with coronary artery event rates.

In the *Nurses' Health Study*, both BMI >25 and physical activity were important predictors of coronary artery disease in 20 years follow up.

Women with a elevated total cholesterol were approximately twice as likely to develop coronary artery disease as those with cholesterol levels of 205mg/dl or

lower. In its recent guidelines, the American Heart Association(AHA) has changed the cut-point for what constitutes low HDL-C in women from less than 40 to less than 50.

In our study, 94% had low HDL-C cholesterol(<50mgt/dl). LDL-C (low density lipoprotein) levels increase with increasing age for both women and men and especially important in women.

The LDL fraction of total cholesterol is a strong predictor of coronary artery disease mortality in women as well as men. Unlike in men whose LDL-C levels plateau at the age of 50 yrs, the LDL-C levels in women increase steadily by average of 2 mg/dl/yr between the age of 40 to 60 (total of 40mg/dl). The optimum LDL-C level is <100mg/dl

Conclusion

The following are conclusions that could be inferred from this study on clinical spectrum and risk factors among female patients.

- The most common symptom is chest pain.
- Atypical symptoms are more common in older females and diabetic patients.
- The most common cardiovascular sign is crackles.
- Most of the patients belonged to Killip Class-I.
- Most common age group affected was above 60yrs and showing that risk of myocardial infarction increases proportionately with age.
- Diabetes is clearly related to risk of myocardial infarction.
- Both obesity and hypertension are also associated with increased risk of myocardial infarction in female population.
- Most of the patients with myocardial infarction have dyslipidemia.
- Most common type of myocardial infarction is anteroseptal myocardial infarction(ASMI)

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PROFORMA:

1. Name

2. Age

3. Marrital status

4. No of Children

5. Educational status

6. Occupation

7. Family Income/yr

8. Address :

9. Hgt in cms

10. Wgt in kgs

11. BMI(kg/M2)

12. Clinical presentation on admission: Chest pain / Nausea / vomiting / Epigastric pain / Jaw pain / Left arm pain / Sweating/ Dyspnoea / Palpitation / Syncope / Others

13. Time of Onset of symtoms :

14. Hypertension : Y / N

How many yrs:

Treatment :

15.Diabetes : Y / N

Type :

Treatment:

16.Hyperlipidemia : Y/N

Treatment:

17. Alcohol : Y /N

18. Smoking: Y / N

19. Family H/O of IHD:

20. Sedentary habits:

21. Menstrual H/O: Premenopausal / Postmenopausal

Menopause: Yrs since menopause

22. Diet H /O : Veg / Non Veg

Frequency

Duration

Type of cooking oil

Salt intake

Coffee/Tea

23. General examination

Conscious: Orientation

BP:

PR:

Anemia:

Cyanosis:

Clubbing:

Icterus:

Pedal edema:

Markers of Hypercholesteremia

24. CVS:

Apical Impulse:

Heart sounds:

Murmurs:

Rub:

25. RS :

Air entry:

Adventitious sounds

26. P /A:

27. CNS:

nvestigations:

1.Urine R /E : Albumin

Sugar

Deposits

2.RFT :Urea

Creatinine

Electrolytes

3.CBC: Total Count Differential Count

Erythrocyte Sedimentation Rate

Hemoglobin Platelets

4. Fasting Blood Sugar

Post Prandial Blood Sugar

5.LIPID PROFILE

6. Serum CK-MB

7. Chest-X-Ray

8. Electrocardiogram:

9. Echocardiogram:

ABBREVIATIONS:

STEMI- ST Elevation Myocardial Infarction

NSTEMI-Non ST Elevation Myocardial Infarction

MI-Myocardial Infarction

BMI-Body Mass Index

CHF-Congestive heart Failure

CAD-Coronary Artery Disease

Wgt-Weight in kilogram

Hgt-Height in centimeter

CP-Chest Pain

Pal-Palpitation

Dys-Dyspnoea

EP-Epigastric Pain

Nau-Nausea

Vomi-Vomiting

Radi-Radiating pain

Oth-Other symptoms

HT-Hypertension

DM-Diabetes Mellitus

KS-Killip score

JVP-Jugular Venous Pressure

PE-Pedal Edema

Al-Alcohol

Smo-Smoking

BP-Blood Pressure

AI Shift-Apical Impulse Shift

Dyslipid-Dyslipidemia

THrombo-Thrombolysis

T-Thrombolysed

N-Not thrombolysed

MR-Mitral Regurgitation

CK-MB-Creatine Kinase

ECG-Electrocardiogram

AWMI-Anterior Wall Myocardial Infarction

ASMI-Antero Septal Myocardial Infarction

LMI-Lateralwall Myocardial Infarction

IWMI-Inferior Wall Myocardial Infarction

PWMI-Posterior Wall Myocardial Infarction

RVMI-Right Ventricular Myocardial Infarction

CKD-Chronic Kidney Disease

LDL-C-Low Density Lipoprotein Cholesterol

HDL-C-High Density Lipoprotein Cholesterol

SLE-Systemic Lupus Erythematosus

S.NO	IP NO.	Age	Wgt	Hgt	BMI	SYMPTOMS								DM
						CP	Pal	Dys	EP	Nau	Vomi	Radi.	oth	
1	27549	54	71	153	30.3	N	Y	Y	N	Y	Y	Y	N	Y
2	27584	60	67	140	34.1	Y	N	N	N	N	N	N	N	N
3	27708	76	55	144	26.5	Y	y	Y	N	N	N	N	N	Y
4	27865	67	56	140	28.6	Y	N	N	Y	Y	Y	Y	N	N
5	27940	68	53	151	23.2	N	Y	Y	N	N	N	N	N	Y
6	28137	76	70	143	34.2	Y	N	Y	N	N	N	N	N	Y
7	28512	61	54	152	23.4	Y	N	N	N	N	N	N	N	N
8	28520	56	58	139	30.0	Y	N	N	N	N	N	N	N	N
9	28580	78	66	141	33.2	N	Y	Y	N	Y	Y	N	N	Y
10	28583	64	56	155	23.3	Y	N	N	N	N	N	N	N	N
11	28586	61	62	146	29.1	Y	N	N	Y	N	N	N	N	Y
12	28733	43	54	143	26.4	Y	y	N	N	N	N	Y	N	N
13	28753	76	47	151	20.6	Y	N	Y	N	Y	N	N	N	Y
14	28882	63	54	141	27.1	Y	N	N	N	N	N	N	N	Y
15	29020	79	63	150	28.0	Y	N	N	N	N	N	N	N	N
16	29109	66	55	147	25.5	N	Y	Y	Y	N	N	N	N	Y
17	29374	58	79	143	38.6	Y	N	N	N	N	N	Y	N	N
18	29439	86	54	148	24.7	Y	N	N	N	Y	Y	N	N	N
19	29529.	67	59	144	28.5	Y	N	N	N	N	N	N	N	N
20	29622	54	53	140	27.0	Y	y	Y	N	N	N	Y	N	Y
21	29789	77	68	142	33.8	Y	N	N	N	N	N	N	N	N

22	30098	53	70	141	35.2	Y	N	Y	N	N	N	N	N	N
23	30115	62	57	142	28.3	Y	Y	N	N	N	N	N	N	Y
24	30240	56	66	138	34.7	Y	N	N	Y	N	N	N	N	N
25	30575	57	45	152	19.5	N	Y	Y	N	N	N	Y	N	N
26	30601	81	46	137	24.5	Y	N	Y	N	N	N	N	N	Y
27	30663	68	69	153	29.5	Y	N	N	N	Y	Y	N	N	N
28	30699	90	70	141	35.2	Y	N	N	N	N	N	N	N	N
29	30725	67	49	139	25.4	N	y	Y	N	N	N	N	N	Y
30	31125	79	63	142	31.2	Y	N	Y	N	N	N	N	N	N

MASTER CHART

S.	IP	Age	Wgt		HBMI	SYMPTOMS	HT				
						CP	Dys	EP	Vomi	Radi	
31	31222	47	52	1	27.3	Y	Y	N	N	N	Y
32	31351	48	59	1	28.1	Y	N	Y	N	Y	N
33	31446	61	59	1	33.4	Y	N	N	N	N	N
34	31548	76	46	1	22.2	N	Y	N	N	N	N
35	31708	65	64	1	34.1	Y	N	N	Y	Y	Y
36	31721	70	57	1	30.4	Y	N	N	N	N	N
37	32059	63	49	1	25.7	N	Y	N	N	N	N
38	32110	55	52	1	28.5	Y	N	Y	N	N	Y
39	32115	68	63	1	36.1	Y	N	N	N	N	N
40	32217	53	61	1	29.0	Y	N	N	N	Y	N
41	32266	77	47	1	23.6	Y	N	N	Y	N	N
42	32438	76	64	1	28.8	N	Y	N	N	N	N
43	32755	52	65	1	36.2	Y	N	N	N	N	Y

44	32783	80	57	1	29.5	N	Y	N	N	Y	N
45	33587	76	61	1	35.6	Y	Y	N	N	N	N
46	33600	48	43	1	19.4	Y	N	N	N	N	N
47	33631	61	59	1	31.0	Y	N	Y	N	N	N
48	33639	44	60	1	33.4	Y	Y	N	Y	N	Y
49	33659	76	54	1	23.7	Y	N	N	N	Y	N
50	33787	62	66	1	30.1	Y	N	N	N	N	N
51	34121	79	59	1	33.4	Y	N	N	N	N	N
52	34481	77	55	1	25.1	N	Y	N	N	N	Y
53	34779	65	67	1	28.3	Y	N	N	N	N	N
54	35034	49	50	1	22.5	Y	N	N	Y	Y	N
55	35237	82	54	1	30.1	N	Y	N	N	N	Y
56	35314	47	77	1	40.4	Y	N	N	N	N	N
57	35695	82	62	1	34.5	N	Y	N	N	Y	N
58	35995	55	64	1	33.1	Y	N	N	N	N	Y
59	36008	79	61	1	29.4	Y	N	Y	Y	N	N
60	36125	63	49	1	24.7	Y	Y	N	N	N	Y

S.no	IP No	Age	Wgt	HBMI	SYMPTOMS	HT				
					CP	Dys	EP	Vomi	Radi	
61	36377	76	60	1	33.9	Y	Y	Y	Y	N	Y
62	36524	46	55	1	24.8	Y	N	N	N	Y	N
63	36563	79	59	1	28.5	Y	N	N	N	N	Y
64	36587	51	52	1	26.5	Y	N	N	N	Y	N
65	36638	61	45	1	19.5	N	Y	N	N	N	Y
66	37117	56	66	1	28.9	Y	N	Y	N	N	N

67	37283	81	70	1	33.3	Y	N	N	Y	N	Y	
68	37441	43	55	1	25.8	Y	N	N	N	N	Y	
69	37471	65	62	1	34.53	Y	Y	N	N	N	N	
70	37726	48	61	1	29.0	Y	N	N	N	Y	Y	
71	37816	90	72	1	39.5	N	Y	N	Y	N	N	
72	37978	42	57	1	24.7	Y	N	N	N	N	Y	
73	38138	64	55	1	28.9	Y	Y	N	N	N	N	
74	38246	70	65	1	30.5	Y	N	N	N	N	N	
75	38506	79	66	1	35.7	N	y	N	N	Y	Y	
76	38675	47	49	1	20.1	Y	Y	Y	N	N	N	
77	38764	77	54	1	27.2	Y	N	N	N	N	N	
78	39008	64	63	1	28	Y	N	N	Y	N	Y	
79	39013	69	60	1	34.96	N	y	N	N	N	N	
80	39127	78	55	1	25.5	Y	N	N	N	Y	N	
81	39386	49	63	1	28.4	Y	Y	N	N	N	Y	
82	39484	80	70	1	35.2	Y	N	N	N	N	N	
83	39603	72	57	1	25	Y	N	N	N	N	Y	
84	39794	65	54	1	30.5	Y	N	N	N	N	N	
85	39812	81	68	1	34.7	N	Y	N	N	Y	Y	
86	39948	51	51	1	27.9	Y	N	N	Y	N	Y	
87	40116	69	62	1	29.5	Y	N	N	N	N	N	
88	40146	76	69	1	33.8	Y	N	N	N	N	Y	
89	40276	68	56	1	25.2	N	Y	Y	N	N	N	
90	40690	54	59	1	30.5	Y	N	N	N	N	N	
S.no	BP	JVP	PE	AI	S3	S4	Creps	Wheeze	Throz	LDL-C	CK-MB	

				shift							
1	150/98	N	N	N	N	N	N	N	T	123	78
2	130/88	Y	N	N	Y	N	Y	N	T	167	87
3	170/98	N	N	N	N	N	N	N	N	148	90
4	120/78	N	Y	Y	N	N	Y	N	N	171	119
5	116/76	N	N	N	N	N	N	N	T	164	104
6	150/100	N	N	N	Y	N	Y	N	T	114	117
7	124/82	N	N	N	N	N	N	N	T	161	79
8	128/80	N	N	N	N	N	Y	Y	N	151	159
9	148/96	Y	Y	N	N	N	N	N	N	174	92
10	128/80	N	N	Y	N	N	N	N	N	179	114
11	80/56	N	N	N	Y	Y	Y	N	T	135	110
12	114/70	N	N	N	N	N	N	N	N	181	117
13	124/76	N	N	N	N	N	Y	N	T	161	120
14	176/108	N	N	N	N	N	N	N	T	109	179
15	132/80	N	N	N	N	N	N	N	N	165	164
16	112/70	N	Y	N	Y	N	Y	N	T	184	123
17	154/94	N	N	N	N	N	N	N	N	139	180
18	126/80	N	N	Y	N	N	Y	N	T	177	163
19	130/78	Y	N	N	N	N	N	N	N	176	98
20	124/78	N	Y	N	N	N	N	N	T	155	131
21	174/108	N	N	N	N	N	Y	Y	T	163	125
22	118/76	N	N	N	N	N	N	N	T	166	147
23	160/102	N	N	N	Y	Y	Y	N	T	119	139

24	126/80	N	Y	N	N	N	N	N	N	168	134
25	124/78	N	N	N	N	N	N	N	T	170	128
26	166/103	N	N	Y	Y	N	Y	N	N	154	190
27	128/80	Y	N	N	N	N	N	N	T	162	94
28	180/100	N	N	N	N	N	N	N	N	98	144
29	130/82	N	N	N	N	N	N	N	N	144	137
30	112/72	N	N	N	Y	N	Y	N	T	169	133
S.no	BP	JVP	PE	A I s h if t	S3	S4	Creps	Whee ze	Thro	LDL-C	CK-MB
31	108/70	N	N	N	Y	N	Y	N	N	167	109
32	168/90	N	Y	N	N	N	N	N	T	154	164
33	124/80	Y	N	Y	N	N	N	N	N	170	113
34	130/80	N	N	N	N	N	Y	Y	N	116	84
35	160/100	N	N	N	N	N	N	N	T	181	86
36	114/78	N	N	N	N	N	N	N	T	164	96
37	84/54	N	Y	N	Y	N	Y	N	T	149	101
38	120/80	N	N	N	N	N	N	N	T	163	156
39	132/82	N	N	N	N	N	N	N	T	102	137
40	150/96	N	N	Y	N	N	Y	N	N	139	113
41	120/84	Y	Y	N	N	N	N	N	N	170	115
42	116/80	N	N	N	N	N	N	N	T	169	98
43	170/104	N	N	N	N	N	N	N	T	131	130
44	124/80	N	N	N	Y	Y	Y	Y	N	175	114
45	130/84	N	N	N	N	N	N	N	T	174	146

46	108/74	N	N	N	N	N	N	N	T	93	121
47	188/100	N	N	N	N	N	N	N	N	176	134
48	126/80	N	Y	N	N	N	Y	N	N	162	140
49	118/80	Y	N	Y	N	N	N	N	N	145	109
50	160/104	N	N	N	N	N	N	N	T	177	114
51	116/76	N	N	N	Y	N	Y	N	T	170	105
52	120/82	N	N	N	N	N	N	N	N	169	151
53	158/98	N	Y	N	N	N	N	N	N	117	135
54	126/74	N	N	N	N	N	N	N	N	174	140
55	124/76	Y	N	N	N	N	N	N	T	165	128
56	170/100	N	N	N	Y	Y	Y	Y	T	134	134
57	116/70	N	N	Y	N	N	N	N	N	166	156
58	124/82	N	N	N	N	N	N	N	N	85	139
59	122/78	Y	Y	N	N	N	Y	N	T	170	109
60	110/70	N	N	N	Y	N	N	N	N	164	103
S.no	BP	JVP	PE	AI shift	S3	S4	Creps	Wheez	Throm	LDL-C	CK-MB
61	150/100	N	Y	N	N	N	N	N	T	134	113
62	114/76	N	N	Y	Y	N	Y	N	T	165	108
63	122/78	N	N	N	N	N	N	N	N	119	159
64	158/96	N	N	N	N	N	N	N	T	161	146
65	112/70	Y	N	N	Y	N	Y	N	N	152	123
66	126/80	N	Y	N	N	N	N	N	N	178	116
67	188/110	N	N	N	N	N	Y	Y	N	169	107

68	120/80	N	N	Y	N	N	N	N	T	142	146
69	110/74	N	N	N	N	N	N	N	N	107	136
70	128/82	N	N	N	Y	N	Y	N	T	133	126
71	178/100	N	Y	N	N	N	N	N	N	181	119
72	136/86	N	N	Y	N	N	N	N	T	146	154
73	130/76	N	Y	N	N	N	N	N	N	166	140
74	148/100	N	N	N	Y	Y	Y	N	T	96	124
75	110/76	N	N	N	N	N	N	N	T	163	139
76	120/70	N	N	N	N	N	N	N	N	136	190
77	130/78	Y	Y	N	Y	N	Y	N	N	174	141
78	150/98	N	N	N	N	N	N	N	N	180	201
79	118/78	N	N	N	N	N	N	N	T	141	125
80	110/76	N	N	Y	N	N	N	N	T	117	78
81	80/60	Y	N	N	Y	N	Y	N	T	168	107
82	122/80	N	N	N	N	N	N	N	N	177	134
83	166/106	N	Y	N	N	N	N	N	N	147	165
84	126/86	N	N	N	Y	N	Y	N	T	101	101
85	120/74	N	N	N	N	N	N	N	T	174	74
86	158/104	N	N	N	N	N	Y	N	T	139	129
87	114/70	N	N	Y	Y	N	Y	Y	N	164	245
88	116/76	N	Y	N	N	N	N	N	T	116	165
89	160/102	N	N	N	N	N	N	N	N	136	141
90	128/78	N	N	N	N	N	Y	N	T	173	115